

aortic crossclamping, avoiding the difficulties related to the injection into the large single coronary ostium. The aortotomy was closed with a double running suture and the aortic crossclamp released.

Recovery of cardiac function was not good; the electrocardiogram showed a left bundle branch block and the left ventricle was dyskinetic. The cardiac movement disorder was more severe when weaning from cardiopulmonary bypass was attempted and high cardiac filling pressures were reached. Retrograde coronary sinus perfusion was used to eliminate a possible gaseous coronary embolus, but it was ineffective. Then we decided to replace the valve prosthesis with a smaller one, to reduce a hypothetical compression of the LADCA and LCCA. When the prosthesis was removed no signs of possible damage or obstruction of the coronary arteries was observed. Blood cardioplegic solution was injected antegradely into the solitary coronary ostium, but no hematoma was visible at the level of the previous sutures. A new 19 mm St. Jude Medical aortic prosthesis was implanted. Unfortunately the situation remained unchanged; weaning from bypass was again unsuccessful because of severe depression of the left ventricle, which remained dyskinetic. Acting on the hypothesis that even the smaller prosthesis, because of its rigid anulus, compressed the anomalous left coronary arteries, we decided at this time to perform a double saphenous bypass graft procedure on the LADCA and the marginal artery of the LCCA. This procedure resolved the situation. Satisfactory cardiac function was soon obtained, even though pharmacologic inotropic support was needed, and the patient was weaned from bypass. Cardiac function quickly improved and the inotropic support was suspended on the first postoperative day. The postoperative recovery was slow as a consequence of the long bypass time, but the patient was discharged from the hospital 3 weeks after the operation. At the 6-month postoperative follow-up examination she was in class I of the New York Heart Association classification, and an echocardiographic assessment revealed normal cardiac function.

In our opinion this case supports the hypothesis that the pathophysiologic mechanism of sudden myocardial ischemia is related to the course of the left coronary artery. When the coronaries arise from the anterior sinus of Valsalva and the left coronary artery passes obliquely between the aorta and the pulmonary artery, an acute angle is formed that can occlude the artery if extrinsic compression is applied. Increased expansion of the aorta and the pulmonary artery during exercise can produce this compression, and sudden death almost always occurs during or immediately after physical activity. In the case of our patient, who had a normal life until the symptoms of the aortic stenosis developed, the rigid prosthetic valvular anulus assumed a relevant part in the described mechanism. Different anatomic relationships between the coronary arteries and the contiguous structures can explain the different outcomes of patients having a similar anomaly. More sophisticated diagnostic tools that could assess exactly this relationship could probably facilitate the choice of a safe operative strategy when the surgical indication is not clear. In the absence of such an oppor-

tunity, only the experience of prior cases can be of help to determine the best treatment for such dramatic situations.

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Primary elective open sternum with only Steri-Drape film coverage after cardiac operations in pediatric patients

To the Editor:

Clear and objective evidence exists that pressure of paracardiac structures on the heart can result in a constricting effect on diastolic ventricular filling.¹ Conversely, relief of the constricting effect results in increased cardiac index and stroke work index, attributable to the Frank-Starling response to increased ventricular preload.¹ Delayed sternal closure to achieve the latter objective after cardiac operations in pediatric patients has been reported.²⁻⁸

A recent article by Hakimi and colleagues⁸ compared the technique of primary open sternum coupled with delayed sternal closure with the use of primary sternal closure in neonates after cardiac operations. The authors mentioned that the techniques for primary elective open sternum with delayed sternal closure can be classified into three broad categories: (1) skin closure with or without sternal stenting, (2) patch closure of the incision with or without sternal stenting, and (3) mediastinal packing. As proponents of the latter technique, Furnary and col-

leagues,⁹ in adult patients, applied sterile laparotomy dressings in the mediastinum and covered the dressings with Steri-Drape plastic film (3M Company, St. Paul, Minn.).

I want to report a modification of the latter technique, namely, covering of the sternotomy wound with Steri-Drape film only. In cases of severe edema of the heart or paracardiac structures, a sternal retractor is left in place. Continuous irrigation of the mediastinum with povidone-iodine (Betadine; Purdue Frederick Company, Norwalk, Conn.) or bacitracin can be performed with substernal irrigation catheters and mediastinal drainage tubes. In case of intractable bleeding, the pericardial space and mediastinum initially can be packed with sterile gauzes or laparotomy pads, which can be removed later as the bleeding subsides. I have applied this technique in seven neonates and infants with severe cardiac edema who had undergone repair of complex congenital cardiac lesions. Delayed closure of the sternum was successfully performed after 2 to 5 days. All patients survived and none had mediastinitis or wound infection.

The main advantages of this technique are as follows: (1) The translucent Steri-Drape film allows continuous assessment of size and functional recovery of the heart; accordingly, it allows secondary sternal closure in a timely fashion; (2) maximal expansion of the pericardial and mediastinal space is obtained by leaving the sternal retractor in place; this cannot be achieved consistently with a technique that involves skin closure or even patch closure; and (3) ease of performance. The Steri-Drape film can be replaced under sterile conditions in the intensive care unit as needed.

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Infections and pathologic factors in the donor lung

To the Editor:

We read with interest the article by Low and associates,¹ titled, "The Donor Lung: Infectious and Pathologic Factors Affecting Outcome in Lung Transplantation." We disagree with a number of the points made in the discussion of the article. Low and associates correctly stated that we treat lung donors with intravenous and aerosolized antibiotics to decrease the prevalence of early bacterial pneumonia in the recipient of lung allografts. This approach is based on experimental data that demonstrate a decreased incidence of pneumonia in the recipient when contaminated donor lungs are treated with this antibiotic combination.² Low and colleagues incorrectly stated that after this regimen was initiated, intrathoracic infection developed in 16 of 37 recipients. This was the incidence of intrathoracic infection in our recipients *before* this regimen was begun. Since we began treating our recipients with combined intravenous and aerosolized antibiotics we have noted a significant decrease in the incidence of early intrathoracic infection and, in particular, a decreased incidence of early bacterial pneumonia. From 1991 to 1993 inclusive, early bacterial pneumonia developed in 11 of 187 recipients (5.9%). In 1993, early bacterial pneumonia developed in one of 68 recipients (1.5%).

The authors also hypothesize: "Treatment of the donors before lung retrieval may temporarily suppress tracheobronchial flora and make the assessment of significant infection at the time of harvest more subject to error." This is not true because appropriate cultures can be taken before donor antibiotic therapy is begun.

We also disagree with the statement: "Proof of invasive infection in donor lungs indicates the need for expedient and specific treatment to avoid progression of the infectious process after implantation." We believe that the presence of invasive infection in a donor lung should be a contraindication to the use of those lungs for transplantation. Indeed, in Low's series, pneumonia developed in